- Difficulty in rising up in the morning.
- By day the patient is sleepy i.e. hypersomnolence, the patient sleeps in the bus, in job, while watching T.V., while driving, and many car accidents are caused by snoring drivers.
- Lack of concentration, stupidity in children.
- Mood and personality changes, e.g. depression.

d) Cardiovascular effects.

Mainly due to hypoxia and CO_2 accumulation during the obstructed breathing and the apneic periods.

1. *Heart rate* is slower, and a dangerous rate may be as low as 40 beats/min.
2. *B. P.* : In normal sleep, the B. P. diminishes by 10- 15% . During the apneic periods, the B. P. rises by 20-30 mmhg , and repetition of the apneas causes a sustained high B. P. Most obese snorers are hypertensive.
- Hypertension resistent to treatment, the G. P. should inquire about sleep, as snoring and OSA may be the cause of resistence to treatment.
3. *Ventricular extrasystoles* may occur during the apneas.
- Patients with ischaemic heart, and those taking medications that increase cardiac irritability are at an increased risk of developing lethal ventricular arrhythmias.
4. *Atrial flutter* and fibrillation may occur and be resistent to treatment.
5. *Pulmonary* hypertension and cor pulmonale.
6. *Sudden death* and coronary attacks, occuring during sleep, always occur near dawn, i.e. at the end of the night, when hypoxia and hypercarbia at their heighest levels.

Investigations :

1. *E.N.T.* specialist is consulted.
2. *Sleep center investigation* :
Where recordings of pulse, B.P, O_2 tension, CO_2 level EEG, ECG,... are made during an 8-hour sleep; this is called polysomnography.
3. *X-rays & fiberoptic* endoscopic examination of the air passages in the upright and supine positions.

Treatment :

1. Weight reduction.
2. Treatment of any nasal and other airway obstructions.
3. U. P. P. P., uvulo palato pharyngoplasty, i.e. resection of the uvula, calculated parts of the soft palate and redundant pillars and pharyngeal mucosa. This usually cures snoring and prevents its bad effects.

7 . EPISTAXIS

This means the escape of blood from the nose.

Causes of epistaxis

A. Local Causes :

1. Idiopathic bleeding from "Little's area" is the commonest cause in adolescence , usually initiated by physical exercise or hot weather.
2. Congenital : e.g. familial telangiectasis on the septum.
3. Trauma : e.g. a blow or compound fracture ; operative trauma ; sharp foreign body.

4. *Inflammatory* : atrophic rhinitis (when the crusts separate), rhinoscleroma, syphilis, or leprosy.

5. *Neoplastic*.

Benign : Angioma ; multiple telangiectasis ; papilloma.

Malignant tumours of the nose.

6. *Deviated septum :* Epistaxis may arise from : a) The convex side (mucosa exposed to the air current, friable and easily bleeds).

b) The concave side (discharge stagnates and froms a crust).

7. Causes in the *nasopharynx :*

Adenoids, nasopharyngeal fibroma, or carcinoma.

8. The nose may be an outlet for bleeding from the inside of the skull, as in fracture base.

B. General Causes :

1. *Arteriosclerosis* ; hypertension ; *puberty* (many girls get epistaxis with the start of menstruation); extreme hot weather ; violent exercise.

2. *High venous pressure* : Mitral stenosis ; emphysema ; mediastinal tumours.

3. *Fevers* : Rheumatic fever. Any rise of temperature in children may be ushered by epistaxis.

4. *Drugs* : Quinine, salicylates, heparin...etc.

5. *Blood diseases* : Haemophilia, leukaemia, purpura, deficiency of vitamin K (e.g. jaundice) or vitamin C (scurvy).

Site of epistaxis :

1. **Little's area** (Kiesselbach's area) is the site in 90% of cases. This is an area on the cartilagenous septum, about 1/4 inch within the vestibule and an equal distance above the floor of the nose (Fig. 22). Here the mucous membrane contains the anastomosis of four vessels :

a) The most important is the sphenopalatine artery which is called the "artery of epistaxis."

b) The anterior and middle ethmoidal vessels.

c) A branch of the greater palatine vessel entering through the incisive foramen.

d) Septal branch of the superior labial (a branch of the facial artery).

2. High up in the nose (anterior ethmoid vessels) especially in hypertension.

3. Other sites : e.g. From a spur on the septum , from the inferior turbinate , or floor of nose.

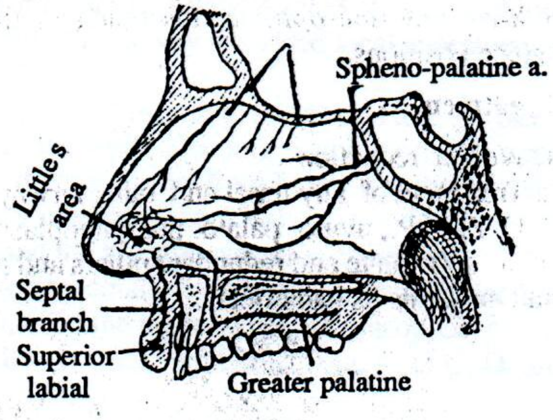

Fig. 22 -Little's area.

Management of epistaxis :

A. Patient history.

- History is obtained from patient or family.
- Ask about first episode -
  which side or bilateral.
  how often bleeding occurs.
  what initiates it : excercise, nose blowing or spontaneous.
- History of trauma, nose or sinus surgery.

- Ask about hypertension , chronic pulmonary disease , liver disease , excess alcohol intake.

- Also ask about drug intake : aspirin, anticoagulant therapy.

B. **First aid measures :**

When epistaxis occurs in a place where medical equipment is not at hand :
- Rest with the head raised. Many people lie flat on their backs during a nosebleed, which causes the blood to drain into the pharynx and results in spitting up blood or swallowing it and vomiting. This creates more anxiety, increasing the blood pressure and the bleeding.
- Cold compresses to forehead and ice to suck.
- Removal of any tight or heavy clothes.
- The patient pinches the tip of the nose, this compresses Little's area and stops most anterior nosebleeds.
- Vasoconstrictors : adrenaline on a piece of cotton packed in the nose.

C. **Management in Hospital**

When the student is confronted with a case of epistaxis in the "Kushuk" the following steps are made in order :

1. Rest the patient on the examination chair, or on the table in the semisitting position. If the patient is shocked the head is lowered and anti-shock measures are undertaken.
2. Order the patient to blow the nose, in order to clean it from blood clots.
3. Pack the nose with pieces of cotton soaked in adrenaline solution and local anaesthetic (pantocaine , N.B. if patient is hypertensive use ephedrine instead of adrenaline). Until the effect appears (about 10 minutes) do some investigations : temperature, pusle, B.P., a sample for complete blood count, bleeding time, coagulation time, prothrombin time ; and if the history is suggestive, liver or renal function tests as well.
4. After 10 minutes remove the pieces of cotton ; if the bleeding has stopped, you can't send the patient home because after a time the V.C. effect will be replaced by rebound V.D. and epistaxis will recur. It is necessary to make a permanent seal to the vessel. This is achieved by cauterization (chemical as chromic acid crystals, AgNo3 50% or trichloracetic acid or electrical). Cauterization is painful , this is why we added local anaesthetic to the adrenaline (see coloured plate - 1 E).
5. If after removing the adrenaline packs there is still bleeding, this means that the bleeding vessel is of some size, and we can't cauterise while bleeding is going on. We apply anterior packing with vaseline gauze. This is done in layers from below upwards applied tightly so as to occlude the bleeding vessels by pressure (Fig. 23 a).
6. After 24 or 48 hours we remove the pack, and the bleeding vessel is cauterized.
7. If a tight anterior pack was put and the bleeding continues this means that the bleeding point is on the posterior border of the septum, or in the postnasal space (nasopharynx). In this case a post nasal pack is done by the E. N. T. specialist (Fig. - 23 C, D and plate - 1 G).
8. *Hypertensive patients.*

Epistaxis, especially in older adults, is frequently associated with hypertension. The arteriosclerotic vessels lying under a delicate mucosa, that is susceptible to drying and cracking , often do not retract and do not clot easily , especially if the B. P. is elevated. Furthermore, the anxiety associated with the epistaxis frequently further elevates the hypertension.

The bleeding usually comes from the anterior and middle ethmoid vessels high up in the nose or from the posterior most part of the septum, and often it is not seen.

If the diastolic blood pressure is markedly elevated, specific antihypertensive management

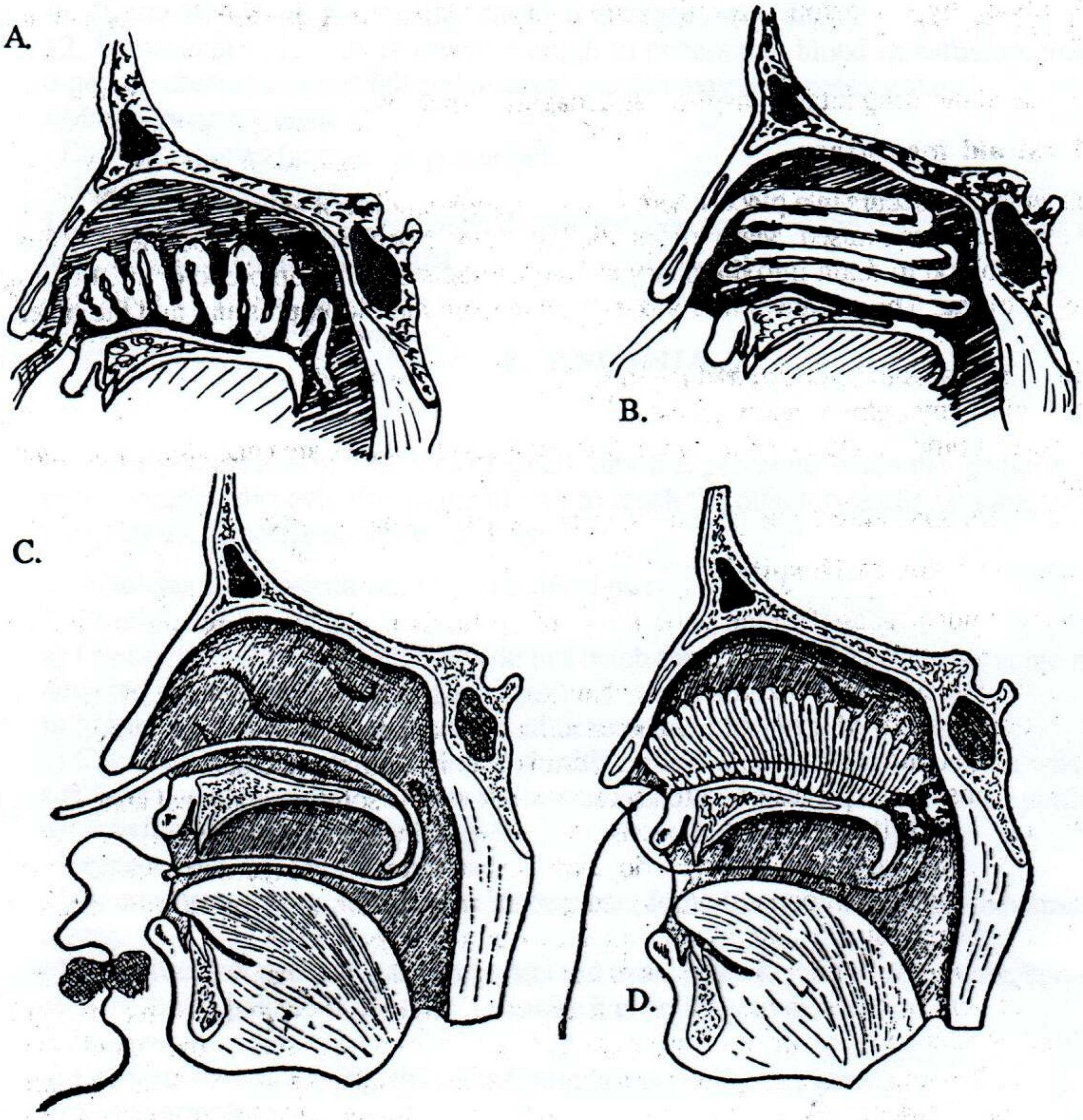

Fig. 23 - Nasal packs for epistaxis.
A . Wrong anterior pack. B . Proper anterior pack.
C . Posterior nasal pack about to be put.
D . Posterior pack in pack in place + anterior pack .

is indicated, but caution not to reduce the B.P. excessively as this may result in cerebral or cardiac infarction.
It is also important not to oversedate a patient who has a nasal packing done, as this may result in serious hypoxemia. Anterior and/or posterior packing is left for a fewdays, during which time antibiotics are given (to avoid sinus or ear complications) together with antihypertensive therapy.
9. Young children with high *fever* , get generalised vasodilation and epistaxis occurs. The whole nose is congested and we can't cauterise all the area, so anterior packing with adrenaline or vaseline gauze, then measures to control the fever. When the temperature subsides, the vasodilataion disappears and bleeding does not recur.
10. ***In children,*** epistaxis is common and usually frightening. Bleeding may follow trauma, nose picking, nose blowing, F.B., muscular execercise, hot weather and spontaneously specially at puberty.

There is often a vessel close to the cutaneous edge of the septum and difficult to anesthesise properly by local anaesthesia, general anaesthesia may be necessary.
11. If granuloma or tumour is the cause, it is treated accordingly.
12. Sometimes epistaxis is severe enough to necessitate blood transfusion, and sometimes the above measures fail and *arterial ligation* may be necessary, e.g.
- Maxillary artery ligation.
- Ethmoid arteries (anterior or posterior).
- External carotid artery.

13. If a bleeding disorder has been diagnosed, correction of the deficient factor is made after advice of the heomatologist.

8. ANOSMIA

Anosmia means loss of the sense of smell. Smell is perceived when the air-borne odoriferous particles dissovle first in the mucus to reach the olfactory endings; impulses hence travel by the olfactory nerves to the brain.

The causes of anosmia may be classified as :
1. Nasal :
a) Obstruction : the smell particles do not reach the olfactory mucosa, e.g. acute rhinitis, deviated septum ; nasal polypi ; new growths.
b) Maldirection of the air current, e.g. after removal of the inferior turbinate.
c) Causes in the mucus : In allergic rhinitis there is excess secretion which washes the smell particles. In atrophic rhinitis the mucus is too thick to dissolve the smell particles.
d) Interference with nerve endings.
- Degeneration, in atrophic rhinitis.
- Irritant vapours : e.g. sulphuric acid, benzene, formaldehyde, and many industrial dusts ... etc.
- Post-influenzal neuritis, has been observed more often in the last few years, some cases recover, but if persists more than 3 months it is unlikely to recover.

2. *Intracranial :*
a) Fracture base involving the cribriform plate and olfactory nerves.
b) Senile atrophy.
c) Tumours of the anterior cranial fossa, e.g. meningioma, neuroblastoma.
3. *Malingering :*
Malingerers will deny being able to detect substances such as ammonia which stimulate the trigeminal nerve.

N.B. Patients with anosmia are at a risk from dangerous fumes and gases and also from fires as they cannot detect the smoke odour.